EPIDEMIC POLIOMYELITIS.*

By PHILIP KING BROWN, M. D., San Francisco.

Historically there was no special importance attached to this disease until its significance as a contageous epidemic disorder was emphasized in recent years. That it has existed endemically but infrequently in all parts of our land was apparent, but that it should become an almost national problem was due solely to the appreciation of its epidemic character. In this role it comes close home to us and especially to you residents of this valley, since the first epidemic of the disease in this state and the third recorded in America occurred in June, 1898, at La Grande, not more than 20 miles distant. To Newmark's service in the San Francisco Polyclinic two of the affected children were brought and from the history obtained it seemed that other similar cases had occurred. As there were on record at that time only 13 definite epidemics of the disease, much interest attached to these few cases, and I went to La Grande to see them. There, in the middle of a hot summer, is an isolated, sun sterilized village of 49 inhabitants four, or one-twelfth of the total number of persons, and one-third of all the children had come down with the disease. One had died. I examined the three survivors—two of them for the second time, and was able to get sufficient history of the fourth case to confirm the diagnosis of poliomyelitis. Since that time there have been epidemics of the disease in this state reported from San Francisco in 1903 by Dr. Alice Wood; in Watsonville and the neighborhood in 1907, reported by me; in Redding and Red Bluff in 1909, also reported by me. In San Francisco in 1910, reported by Dr. C. E. Fleischner. In Palo Alto in 1911, reported by Dr. R. L. Wilbur and in the most unsanitary suburb of Los Angeles in 1912, by Dr. T. J. Orbison. The striking characteristics of these epidemics have been their generally rural or suburban character where the sanitary conditions were poor or where distinctly unsanitary conditions were the rule.

In collecting data regarding the disease several unrecorded epidemics in other states were uncovered, notably one in Idaho in 1902 in which some 25 children were attacked.

A brief résumé of the epidemics is given to show the increasing recognition of the disease, or its spread, or both:

Epidemics.

1st record—Colmer. Am. Jr. Med. Sci. July, 1843, 8 or 10 cases in 1841—W. Feliciana, La.

2nd record—Bergenholtz. Mentioned by Marie. 13 cases, 1881—Umea.

3rd record—Cordier, Lyon Medical, 1888. 13 cases, 1885—Lyon, France.

Then began the more general recognition of the epidemic character of the disease in Norway, Sweden, Germany, Italy, S. Australia, New England and finally its occurrence in hundreds of cases in single localities, chiefly in and around Boston, New York, and in Nebraska, and Minnesota, Norway and Oceania.

Epidemics recorded in 5-year periods from 1880:

	Epidemics.	Cases	Average-number cases	by decades			
1880-84	2	23	11.5	1880-89	9	116	13
1885-89	7	93	13				
1890-94	4	151	38	1890-99	27	496	18
1895-99	23	345	15				
1900-04	9	349	39	1900-09	34	8403	280
1905-09	25	8,054	322				

Over 5000 of the cases reported in the last five year period occurred in the United States, and in the following year, 1910, it is estimated that the total reached fully 3000.

The recent Los Angeles epidemic briefly reported by Orbison (*Cal. State Jr.*, Oct., 1912), is a splendid example of proper handling of what might have been a most alarming spread of the disease. In the middle of June a few cases were reported in an outlying and neglected district of the city along both sides of the river bed "where many of the city streets had never been watered and where the hygienic conditions of the inhabitants were bad."

As soon as the epidemic nature of the trouble was fully appreciated the mayor called a meeting of physicians, clergymen and laymen; hospital and publicity committees were named, a suitably located building was promptly put in order and equipped for an isolation hospital, the city council providing funds and moral support, enabling the committee to provide also strict quarantine. The district was cleaned up—all streets were oiled or watered and the result was that the number of new cases fell rapidly so that whereas 41 were reported the last week in July only two were reported the last week in August.

There will always be doubters who tell you that this sort of handling of epidemics is unnecessary and costly, it hurts trade, the publicity it makes is harmful to municipal growth. These are the selfish and superficial critics who fail to hear the question asked, how does the community meet its dangers; does it hide them and seek to prevent any knowledge of them from going abroad, or has it met them skilfully and promptly and whole-heartedly? The people are suspicious of concealment and they can be educated easily to appreciate modern measures of prevention of disease. Even Senator Works, in a recent address in San Francisco, after getting his audience laughing over extracts from a State Health Bulletin recommending protection of latreens from flies because typhoid was at times a fly-borne disease (the very idea!), pasteurizing milk to prevent infantile digestive disorders (he never was raised on anything but good condensed milk, or cow's milk from a good old cow), was obliged to explain when they roared after he read in the next paragraph,—that mosquitoes transmitted malaria and yellow fever,—that this really was true, he knew it from personal investigation in Washington. Thank God for that much light in our national representative; and it does show the possibilities of education.

Causes. So far all that we know is based on a study of seasonal relation of epidemics in which

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it is shown that the disease is commonest in summer, but it may occur in any season. This is especially significant when it was shown that the channel of infection is through the tonsil or nasopharyngeal mucous membranes and that a communicable virus has been recovered as late as six months after recovery from the acute symptoms of the disease. The virus passes readily through a Berkefeld filter and is unchanged by drying, freezing or suspension in glycerine. It has never been cultivated and it is ultra microscopic. In this respect it belongs in the class with the virus of rabies, dengue, scarlet fever, mumps and measles.

Contageousness. Zappert is almost alone amongst recent writers in opposing the idea of contageousness of the disease. Marie of the earlier French writers, including Charcot, laid great stress on the hereditary nature of the disease, pointing to the known occurrence of the disease in several members of one family as evidence of hereditary influences. Emerson shows that 166 children were exposed in the Colrain epidemic in Deerfield Valley, in families in which 67 cases occurred and only four of the 166 contracted the disease. There were 16 cases where children slept with a brother or sister having the disease and 21 more where personal contact was intimate and only two of these got the disease. Morse reports that no case ever developed in the wards of the Children's Hospital in Boston although patients with the disease have been treated there for years, nor did the introduction of a case (presumably acute) into St. Mary's Infant Asylum in 1909 result in any contagion.

On the other hand Wickman's report of the 1905 epidemic in Norway shows contact as the likely factor and traces the spread from village to village by contacts. Harbitz of Christiania concurs in this opinion and reports cases among nurses caring for acute cases. Flexner calls attention to carriers, in his suggestion that the big epidemics in this country occurred first in the seaports and then in the middle west, especially the parts settled by Scandinavians, among whom most of the European epidemics have occurred.

The Nebraska epidemic in 1910 (Shidler, *Jr. A. M. A.*, Jan. 22, 1910; McClanahan, *Jr. A. M. A.*, Oct. 1, 1910; Anderson, *Jr. A. M. A.*) shows the rural occurrence but no conclusive data on exposure to a common source of the virus or personal contact as the underlying factor in the spread of the disease. Shidler reports four and six cases in two families of six children, four in a family of five, and four in another of four children, but the vast majority of all cases occurred singly and over widely scattered territory.

Like all epidemic diseases the varying intensity in virulence plays an important role. By passing the virus through 20 monkeys Flexner has secured a potency which practically kills all monkeys inoculated. In studies of experimental transmissibility many domestic animals, chickens and pigeons, have resisted infection, but in the Vermont epidemic (Claverly) spinal cord changes were found in the chickens (Dana) and dogs, horses and pigs were attacked. Chickens were attacked in the Michigan epidemic of 1907 and Westphalia epidemic in 1908. The interesting fact has been established that the disease cannot be transmitted in monkeys through preparations of urine or bile of the affected animals. However, it may be transmitted by emulsions from nasal mucous membranes and intestinal discharges of monkeys intradurally inoculated, hence the nasal and mouth secretions and the feces are dangerous.

Theobald Smith has recently shown that certain of the paralyses of animals were probably unrelated to epidemic poliomyelitis, because emulsions of the spinal cord of recently paralyzed animals inoculated into monkeys failed to reproduce the disease. Paralysis in animals, however, is known to occur from a number of causes and it would seem that safe conclusions as to the disease being primarily one in animals cannot be drawn without repeated inoculation experiments with animals paralyzed during epidemics of the disease among human beings. In the meantime the Massachusetts State Board of Health (1911) reports paralysis among pigs preceding the epidemic at Woburn among human beings; symptoms in cows preceded it in Newton and in a cat in Lowell.

The recent studies of Richardson, Sheppard, Brues and Rosenau, all of Massachusetts, point strongly to the relation of the common biting horse fly (stomoxys calcitrans) to the disease. Richardson in 1911 called attention to the fact that this fly was the only insect constantly present in the majority of houses where the disease had occurred. Brues and Sheppard carefully studied the conditions surrounding 88 cases of the disease in seventeen towns in Massachusetts. They concluded that "fly-time" marked the advent and spread of the disease which suggested the innocence of such insects as fleas, etc., which are less periodic in their appearance. Furthermore in towns where the disease occurred, unsanitary conditions, particularly those inviting fly breeding, were constantly present and the percentage of domestic animals, cows, pigs, horses, etc., averaged 5 to 20 times higher per human inhabitant than in towns where the disease had not occurred. It is interesting in this connection to recall the fact that in Norway and Sweden where so many of the extensive epidemics have occurred, the family stable and cow barn is an integral part of the house and conditions make for more than usually close contact of human beings and animals.

Finally, Rosenau showed that well monkeys put in cages with horse flies which had been allowed to bite other monkeys with the disease contracted a condition similar to the affected monkeys.

Our own Board of Health are preparing to repeat these experiments. In the meantime it is reasonably certain that the disease is also transmitted directly by acute cases and by carriers.

Incubation. The period has been variously estimated at from 24 hours to several weeks. It is probably very short, the variation being due to the length of time that the virus remains latent in the nasopharyngeal mucous membranes.

Immunity. Animals recovering from the disease produced by inoculation have shown a definite re-

sistance to re-inoculation (Flexner) but Stephens (*Intercolonial Med. Jr.,* Australia, 1908), and Eschner (*Med. Record,* Sept., 1910), report possible recurrences of the disease in the same patient.

No important evidences of antigen or antibodies have as yet been shown in the spinal fluid or serum.

Symptoms. When it is remembered that any part of the nervous system, brain, spinal cord, or nerves may be the seat of the trouble the variation of clinical type is better understood. Wickman divides these clinical types into eight forms, according to the location of the trouble in the nervous system. Of course combinations of types are common.

1. Spinal poliomyelitic.
2. Ascending form (less often descending) Landry's paralysis.
3. Bulbar poutine form.
4. Cerebral or encephalitic form.
5. Ataxic.
6. Polyneuritic.
7. Meningitic.
8. Abortive, in which no paralysis occurs but rather symptoms of meningeal, gastro-intestinal or general infection, hyperesthesia and pain.

These cases must all escape recognition except in epidemics, where according to Wickman they number from 15% to 50%. Frost in the Massachusetts and Iowa epidemics thought them 25% to 50% of the total cases. Anderson, 40% in the Nebraska epidemic, while Müller records an epidemic in Nauru, Oceania, of 700 cases where only 50 showed paralysis after three months.

As paralysis is not necessarily a symptom of the disease and may be so fleeting when present as to be of small consequence, we must first consider the prodromal symptoms and those present in these abortive cases. Hyperesthesia, irritability and sweating have marked the onset of most cases, fever being undoubtedly present at some time during the attack, however brief. It may reach 104° very soon after onset. There is often headache, joint pain, photophobia, constipation or diarrhea, retention or incontinence of urine, vertigo, choreiform movements, twitchings, convulsions, tremor and even coma. Skin eruptions are common in some epidemics and present a varying character. Lovett, Meyer and Strumpell report acute poliomyelitis in association with acute exanthemata, the latter reporting encephalitic form especially after measles. I myself have seen one possible similar case.

In the more serious cases paralysis intervenes and may occur in any part of the body. In these cases particularly the reflexes are important.

In early stages (irritative) the knee and ankle reflexes may be exaggerated but rapidly disappear, not necessarily at the same time. One leg, for example, may be spastic with exaggerated reflexes and the other flaccid with no knee reflex.

In pyramidal involvement a Babinski may occur and even ankle clonus.

Koenig's sign is of doubtful value, being often a varying condition in the rapidly changing state of the central lesions. The neck rigidity may be marked or absolutely wanting, the superficial reflexes present or absent or unequally affected in the same case.

Blood shows a leukocytosis as a rule and this may reach 34,000 (Morse).

Spinal fluid may be slightly turbid, but is clear as a rule. There are increased numbers of cells, chiefly lymphocytes in the later stage where the differential count shows a picture similar to tubercular or syphilitic meningitis. In the early stages the count may show a predominance of polynuclear forms which is exceedingly misleading.

Treatment; prophylaxis. It has been shown that the virus enters the body commonly through the air passages and is readily destroyed by such solutions as may be used harmlessly in these passages. Flexner in a verbal communication to the Association of American Physicians in 1911 recommends the use of mild alkaline sprays and menthol. The now well known fact that hexamethylenamine administered by mouth or in solution by rectum results in the appearance of formalin in the secretions and specially in the spinal fluid in a few minutes, offers the best reason for prompt administration of sufficient doses of this drug in suspected cases and as a prophylactic in exposed cases. Apart from a continuance of these remedies once the disease has established itself, the treatment in an acute stage is symptomatic. Remembering the skin eruptions and the changes noted in the liver, kidneys and spleen, attention must be paid to elimination and the state of the gastro-intestinal tract. I cannot believe Morse is right in the statement that after the paralysis hexamethylenamine does no good, "because the harm has then already been done." The best authorities, Wollstein, Flexner, Gay, Lucas and others have failed to find evidence of antigen in the spinal fluid of monkeys or human beings in various stages of the disease, or of antibodies in the blood serum of monkeys in acute stages of the disease, it seems reasonable to suppose that urotropin cannot interfere with nature's curative process and may limit the infection and its spread. If administered under supervision it can do no harm. Williams is authority for the statement that it "completely failed to arrest the inflammation in cord and meninges, although thoroughly tested in the Washington, D. C., epidemic this year" (1910). He fails to define "thoroughly," however, and the progress of symptoms from the cord involvement is dependent largely on local hyperemia and inflammatory edema so that his deductions seem badly taken. Until there is reason for not giving it, there seems no reason for its discontinuance.

Williams cites a case of the ascending form in which progress ceased after lumbar puncture and 1/3-1/4 gr. bichloride of mercury administered by hypodermic, five doses in three days. There are no reports of the benefits of injections of arsenical derivations, but in Elrich's summary of the benefits of salvarsan he shows its possible benefits in various acute infections, including malaria and smallpox.

In regard to the possibilities of an eventual serum treatment Flexner says, "it cannot be predicted how soon or whether ever at all such a form of specific treatment will be applicable."

The use of electricity and strychnia in the acute stage is obviously bad. Warm baths, splints for resting painful joints, protection from pressure of bed clothing for hyperesthesia are among the rational indications. To the orthopedist finally will go the majority of cases for resulting paralysis and the supervision of these cannot begin too soon.

Prognosis. Death occurs in 5% to 10% of severe cases—probably less than 5% of all abortive cases are recognized. Paralysis occurs in at least 50% to 90% of all reported cases and here, too, it may be that the smaller percentage was in epidemics where abortive cases were so frequently overlooked.

Paralysis, however bad at the beginning, may clear up entirely. Improvement generally takes place for six months, but no change for the better can be looked for after a year. Slightly paralyzed cases may not improve at all.

Resumé. What we know as acute anterior poliomyelitis is an acute infectious disease of unknown origin, occurring of late years in epidemics, small as compared to typhoid, diphtheria and scarlet fever epidemics but of about equal mortality. Like scarlet fever its virulence varies and while it is endemic in this state it rarely shows a strikingly virulent contageous tendency.

It is marked in abortive type by symptoms resembling nasal and tonsilar infection, with muscle, skin and joint pain, by gastro-intestinal symptoms, and finally in paralytic cases by flaccid paralysis of varying muscles in any part of the body.

Its recognition in the prodromal stage in the abortive type is almost impossible except in epidemics.

There is no specific treatment.

THE DIAGNOSIS OF TUBERCULOSIS OF THE SKIN.*

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In the diagnosis of tuberculosis of the skin, we are confronted with a difficulty that we do not have to contend with in the gross pathological conditions of the same process in other organs of the body, inasmuch as we have, in tuberculosis of the skin, a variety of lesions which differ widely clinically, depending on the duration, intensity and evolution of the process, as well as the results of treatment. Not only in the clinical appearance does this wide variation occur, but also in the histological picture do we find the greatest latitude. On one side we will find cases in which the cutis and subcuticular tissue are thickly beset with typical tubercles, surrounded by inflammatory infiltration, with not infrequent tubercle bacilli; while, in other cases, we can only find an isolated tubercle, surrounded by fibrous tissue, and the most energetic search will not enable us to find a single bacillus. Since histopathological sections, showing the typical structure of the tubercle, are not a definite indication of tuberculosis, as similar infiltrations, showing giant cells, are found in other pathological entities, it has often been impossible to make a definite diagnosis from a biopsy, unless the causative agent, that is, the tubercle bacillus, can be found.

Were every case, clinically and histopathologically, a typical one, there would be no difficulty in making a definite diagnosis, but, unfortunately, the picture varies considerably both in the microscope and in gross appearance, and it is in the border line cases that we must exercise every possible endeavor to classify the lesion.

Were it possible, in every case, to find the tubercle bacillus, the diagnosis would be easy, but, unfortunately, it is often impossible to demonstrate the organism, and when found, to state the fact mildly, the number is exceedingly few. In fact, owing to the great difficulties presented in finding the bacillus, which was probably due to the insufficiency of our methods of investigation, it was seriously questioned whether or not certain polymorphous forms of atypical skin lesions, in reality tubercular in origin, were due to the tubercle bacillus, per se, or due to toxins, originating in a tuberculous process elsewhere in the body, and circulating in the blood; and, on this basis, these lesions were designated tuberculides or toxi-tuberculides.

The paucity of tubercle bacilli in skin lesions is probably due to the comparatively small amount of vascularization of the skin and the large amount of connective tissue present, which furnishes a much poorer medium for growth than the parenchyma of the lungs and other highly vascularized organs. This can be well demonstrated by the increased rapidity of progress that takes place when a comparatively indolent form of lupus vulgaris passes over from the skin to the mucous membrane.

However, in consequence of our knowledge of newer methods and better technic in the search for tubercle bacilli, we find that many clinical entities, some formerly classed as tuberculides, and some, where even the designation of tuberculide was disputed, are definitely due to the tubercle bacillus, and we can demonstrate the organism therein.

The methods employed in the diagnosis of tuberculosis of the skin may be classified as follows:

1. Clinical.
2. Animal inoculation.
3. Tuberculin test.
4. Histopathological.
5. Tinctorial.

The clinical appearances of the various tuberculous lesions can hardly be entered upon in a paper of this character; they differ, in their various manifestations exceedingly, but all have the characteristics of torpidity, infiltration, ulceration, and are not infrequently accompanied by other tuberculous lesions.

Animal inoculation, when positive, is practically certain, but, owing to the sparcity of the organism, or the lack of vitality thereof, or some unknown reason, it is not easy to procure positive results, except in the most pronounced cases, which can usually be recognized clinically. Furthermore, the

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